

09/732,360.

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(metabolic pathway and GA binding protein)and (microcompetition) and diseases	0

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Database: IBM Technical Disclosure Bulletins

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protein)and (microcompetition) and
diseases

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<u>DB Name</u>	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u>
USPT	(metabolic pathway and GA binding protein)and (microcompetition) and diseases	0	<u>L3</u>
USPT	(metabolic pathway and GA binding protein)and diseases	68000	<u>L2</u>
USPT	(metabolic pathway and GA binding protein)	224332	<u>L1</u>

```
=> s metabolic pathway
    157412 METABOLIC
      9 METABOLICS
    157416 METABOLIC
        (METABOLIC OR METABOLICS)
    128587 PATHWAY
    131568 PATHWAYS
    235501 PATHWAY
        (PATHWAY OR PATHWAYS)
L1      6310 METABOLIC PATHWAY
        (METABOLIC (W) PATHWAY)

=> s GA binding protein
    6061 GA
    114036 GAS
    119865 GA
        (GA OR GAS)
    564198 BINDING
    1137 BINDINGS
    564449 BINDING
        (BINDING OR BINDINGS)
    1030658 PROTEIN
    869837 PROTEINS
    1341427 PROTEIN
        (PROTEIN OR PROTEINS)
L2      65 GA BINDING PROTEIN
        (GA (W) BINDING (W) PROTEIN)

=> s 11 and 12
L3      0 L1 AND L2

=> s 12 and risk factor
    475338 RISK
    51648 RISKS
    497162 RISK
        (RISK OR RISKS)
    511281 FACTOR
    1429669 FACTORS
    1762656 FACTOR
        (FACTOR OR FACTORS)
    225852 RISK FACTOR
        (RISK (W) FACTOR)
L4      0 L2 AND RISK FACTOR

=> s cellular microcompettion
    244936 CELLULAR
      2 CELLULARS
    244937 CELLULAR
        (CELLULAR OR CELLULARS)
    0 MICROCOMPETTION
L5      0 CELLULAR MICROCOMPETTION
        (CELLULAR (W) MICROCOMPETTION)

=>
```

732,260

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s GABP metabolic pathways
125 GABP
1 GABPS
125 GABP
(GABP OR GABPS)
156905 METABOLIC
9 METABOLICS
156909 METABOLIC
(METABOLIC OR METABOLICS)
130947 PATHWAYS
L1 0 GABP METABOLIC PATHWAYS
(GABP (W) METABOLIC (W) PATHWAYS)

=> s GABP
125 GABP
1 GABPS
L2 125 GABP
(GABP OR GABPS)

=> s disruption and l2
32869 DISRUPTION
2482 DISRUPTIONS
34790 DISRUPTION
(DISRUPTION OR DISRUPTIONS)
L3 3 DISRUPTION AND L2

=> d l3 ibib abs total

L3 ANSWER 1 OF 3 MEDLINE
ACCESSION NUMBER: 1998343943 MEDLINE
DOCUMENT NUMBER: 98343943 PubMed ID: 9677314
TITLE: 4-Aminobutyrate (GABA) transporters from the
amine-polyamine-choline superfamily: substrate specificity
and ligand recognition profile of the 4-aminobutyrate
permease from Bacillus subtilis.
AUTHOR: Brechtel C E; King S C
CORPORATE SOURCE: Department of Physiology and Biophysics, University of
Texas Medical Branch, Galveston, Texas 77555-0641, USA.
CONTRACT NUMBER: T32-ES07254 (NIEHS)
SOURCE: BIOCHEMICAL JOURNAL, (1998 Aug 1) 333 (Pt 3) 565-71.
Journal code: 9YO; 2984726R. ISSN: 0264-6021.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199809
ENTRY DATE: Entered STN: 19981006
Last Updated on STN: 19981006
Entered Medline: 19980923

AB A previous study [Ferson, Wray and Fisher (1996) Mol. Microbiol. 22, 693-701] has shown that transposon-mediated **disruption** of a protein 47% identical to the Escherichia coli GABA (4-aminobutyrate) transporter abolishes the ability of nitrogen-limited culture conditions to induce expression of a GABA transport activity in Bacillus subtilis. Here it is demonstrated directly that the B. subtilis GABA permease (**gabP**) gene can complement the transport defect in the **gabP** -negative E. coli strain. Unexpectedly, the ligand-recognition profile of the B. subtilis **GabP** was found to differ substantially from that of the highly homologous E. coli **GabP**. Unlike the E. coli **GabP**, the B. subtilis **GabP**: (i) exhibits approx. equal preference for the 3-carbon (beta-alanine, Km=9.6 microM) and the 4-carbon (GABA, Km=37 microM) amino acids, and (ii) resists inhibition by bulky,

conformationally constrained compounds (e.g. nipecotic acid, guvacine); which are active against GABA transporters from brain. The present study shows additionally that the *B. subtilis* **GabP** can translocate several open-chain GABA analogues (3-aminobutyrate, 3-aminopropanoate, cis-4-aminobutenoate) across the membrane via counterflow against [3H]GABA. Thus, consistent with the idea that the ligand-recognition domain of the *B. subtilis* **GabP** is less spacious than that of the close homologue from *E. coli*, the former exhibits more stringent requirements than the latter for substrate recognition and translocation. These distinct functional characteristics of the *E. coli* and *B. subtilis* GABA transporters provide a basis by which to identify ligand-recognition domains within the amine-polyamine-choline transporter superfamily.

L3 ANSWER 2 OF 3 MEDLINE
 ACCESSION NUMBER: 97439460 MEDLINE
 DOCUMENT NUMBER: 97439460 PubMed ID: 9295016
 TITLE: PU.1 negatively regulates the CD11c integrin gene promoter through recognition of the major transcriptional start site.
 AUTHOR: Lopez-Rodriguez C; Corbi A L
 CORPORATE SOURCE: Hospital de la Princesa, Madrid, Spain.
 SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (1997 Aug) 27 (8) 1843-7. Journal code: EN5; 1273201. ISSN: 0014-2980.
 PUB. COUNTRY: GERMANY; Germany, Federal Republic of Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199709
 ENTRY DATE: Entered STN: 19971013
 Last Updated on STN: 19971013
 Entered Medline: 19970930

AB CD11c integrin expression is restricted to myeloid cells and activated B lymphocytes, mainly through the collaborative action of Sp1 and members of the AP-1 and C/EBP transcription factor families on the proximal region of the CD11c gene promoter. While analyzing the role of an initiator-like sequence at the major transcriptional start site, an inverted consensus GGAA Ets binding site was identified as a negative regulatory element whose **disruption** increases the activity of the CD11c promoter. The GGAA element was specifically recognized by PU.1 in THP-1 monocytic cells and by PU.1 and **GABP**-related proteins in U937 promonocytic cells. Mutational analysis indicated that PU.1 recognition depends not only on the GGAA consensus element but also on flanking sequences. The functional relevance of PU.1 binding was assayed in transactivation experiments in HeLa cells, where PU.1 co-expression led to a significant decrease in the activity of the CD11c promoter, demonstrating that PU.1 inhibits the activity of the CD11c promoter through a PU.1 binding site located at the major transcriptional start site (PU1-5). The inhibitory action of PU.1 on CD11c is in contrast with its positive regulatory effect on the CD11b and CD18 integrin gene promoters, which might contribute to the differentially regulated expression of CD11b/CD18 and CD11c/CD18 during monocyte extravasation and terminal maturation. In addition, since PU.1 transcriptional activity correlates with macrophage proliferation, PU.1 might modulate CD11c gene transcription according to the proliferative state of the cell.

L3 ANSWER 3 OF 3 MEDLINE
 ACCESSION NUMBER: 96007509 MEDLINE
 DOCUMENT NUMBER: 96007509 PubMed ID: 7559529
 TITLE: **GABP** and PU.1 compete for binding, yet cooperate to increase CD18 (beta 2 leukocyte integrin) transcription.
 AUTHOR: Rosmarin A G; Caprio D G; Kirsch D G; Handa H; Simkevich C P

CORPORATE SOURCE: Brown University School of Medicine, Division of
Hematology, Miriam Hospital, Providence, Rhode Island
02906, USA.

CONTRACT NUMBER: R29 DK 44728 (NIDDK)

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Oct 6) 270 (40)
23627-33.
Journal code: HIV; 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199511

ENTRY DATE: Entered STN: 19951227
Last Updated on STN: 19951227
Entered Medline: 19951114

AB CD18 (beta 2 leukocyte integrin) is a leukocyte-specific adhesion molecule that plays a crucial role in immune and inflammatory responses. A 79-nucleotide fragment of the CD18 promoter is sufficient to direct myeloid transcription. The CD18 promoter is bound by the B lymphocyte- and myeloid-restricted ets factor, PU.1, and **disruption** of the PU.1-binding sites significantly reduces promoter activity. However, PU.1 alone cannot fully account for the leukocyte-specific and myeloid-inducible transcription of CD18. We identified a ubiquitously expressed nuclear protein complex of extremely low electrophoretic mobility that also binds to this region of the CD18 promoter. This binding complex is a heterotetramer composed of **GABP** alpha, and ets factor, and **GABP** beta, a subunit with homology to Drosophila Notch. **GABP** alpha competes with the lineage restricted factor, PU.1, for the same critical CD18 ets sites. The CD18 promoter is activated in myeloid cells by transfection with both **GABP** alpha and **GABP** beta together, but not by either factor alone. Transfection of non-hematopoietic cells with the two **GABP** subunits together with PU.1 is sufficient to activate CD18 transcription in otherwise non-permissive cells. Thus, **GABP** and PU.1 compete for the same binding sites but cooperate to activate CD18 transcription.

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L4 11 L3

=>

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PROCESSING COMPLETED FOR L4
L5          3 DUP REM L4 (8 DUPLICATES REMOVED)
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              AB, NCL, CC, BC, IT (CT, ST), CO, NA, GT, ORGN, RN
BIB ----- AN, DN, TI, AU, CS, PI, SO, NTE, DT, FS, LA, SL
CBIB ----- AN, compressed bibliographic information
DALL ----- ALL, delimited for post-processing
IABS ----- ABS, with text label
IALL ----- ALL, indented with text labels
IBIB ----- BIB, indented with text labels
IIND ----- IND, indented with text labels
IND ----- NCL, CC, BC, IT (CT, ST), CO, NA, NP, GT, ORGN, RN

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HITIND ----- IND
KWIC ----- Hit terms plus 20 words on either side
OCC ----- number of occurrences of hit terms and fields
              in which they occur
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s human GABP
  7503616 HUMAN
  97087 HUMANS
  7522518 HUMAN
    (HUMAN OR HUMANS)
    125 GABP
      1 GABPS
    125 GABP
      (GABP OR GABPS)
L1      1 HUMAN GABP
      (HUMAN(W) GABP)

=> s cellular microcompetition
  244275 CELLULAR
    2 CELLULARS
  244276 CELLULAR
    (CELLULAR OR CELLULARS)
    0 MICROCOMPETITION
L2      0 CELLULAR MICROCOMPETITION
      (CELLULAR(W) MICROCOMPETITION)

=> s microcompetition and human diseases
  0 MICROCOMPETITION
  7503616 HUMAN
  97087 HUMANS
  7522518 HUMAN
    (HUMAN OR HUMANS)
  1305724 DISEASES
    2466 HUMAN DISEASES
      (HUMAN(W) DISEASES)
L3      0 MICROCOMPETITION AND HUMAN DISEASES

=> s l1 and diseases
  1305724 DISEASES
L4      0 L1 AND DISEASES

=>
```

=> s GABP

125 GABP
1 GABPS

L5 125 GABP
(GABP OR GABPS)

=> s diseases

L6 1305724 DISEASES

=> s 15 and 16

L7 1 L5 AND L6

=> d 17 ibib abs

L7 ANSWER 1 OF 1

MEDLINE

ACCESSION NUMBER: 2000087178 MEDLINE

DOCUMENT NUMBER: 20087178 PubMed ID: 10618716

TITLE: Ets transcription factors cooperate with Sp1 to activate the human tenascin-C promoter.

AUTHOR: Shirasaki F; Makhluf H A; LeRoy C; Watson D K; Trojanowska M

CORPORATE SOURCE: Department of Medicine, Division of Rheumatology, Hollings Cancer Center, Medical University of South Carolina, Charleston, South Carolina, SC 29425-2229, USA.

CONTRACT NUMBER: AR42334 (NIAMS)

SOURCE: ONCOGENE, (1999 Dec 16) 18 (54) 7755-64.
Journal code: ONC; 8711562. ISSN: 0950-9232.

PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200002

ENTRY DATE: Entered STN: 20000218
Last Updated on STN: 20000218
Entered Medline: 20000204

AB Tenascin-C (TN-C), an extracellular matrix glycoprotein is expressed during embryonic development, but is present only at low levels in normal adult tissues. TN-C is re-expressed during wound healing, fibrotic **diseases** and in cancer. To better understand the mechanisms that control TN-C gene expression, we examined the regulation of the human TN-C promoter in human fibroblasts. We demonstrate that a short segment of the TN-C promoter between bp -133 and -27 contains three evolutionarily conserved Ets binding sites (EBS). These three EBSs bind in vitro expressed Flil protein and mediate transactivation of the TN-C gene by Flil. Furthermore, two proximal EBSs contribute significantly to basal activity of the TN-C promoter. **GABP**, which is present in human fibroblast nuclear extracts, interacts with the two proximal EBSs. In addition, several Sp1 and Sp3 binding sites have been located in close proximity to the EBSs within this promoter region. The studies performed in Drosophila cells demonstrate that either Flil or GABPalphabeta1 functionally interact with Sp1 resulting in a synergistic stimulation of the TN-C promoter activity. In conclusion, this study shows for the first time that the TN-C gene is regulated by Ets proteins, which together with Sp1 act as potent activators of TN-C expression.

=>

1 ANSWER 1 OF 1 MEDLINE
 ACCESSION NUMBER: 97169219 MEDLINE
 DOCUMENT NUMBER: 97169219 PubMed ID: 9016666
 TITLE: Functional domains of transcription factor hGABP
 betal/E4TF1-53 required for nuclear localization and
 transcription activation.
 AUTHOR: Sawa C; Goto M; Suzuki F; Watanabe H; Sawada J; Handa H
 CORPORATE SOURCE: Faculty of Bioscience and Biotechnology, Tokyo Institute of
 Technology, Midori-ku, Yokohama, Japan.
 SOURCE: NUCLEIC ACIDS RESEARCH, (1996 Dec 15) 24 (24) 4954-61.
 Journal code: O8L; 0411011. ISSN: 0305-1048.
 PUB. COUNTRY: ENGLAND: United Kingdom
 Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199703
 ENTRY DATE: Entered STN: 19970321
 Last Updated on STN: 19970321
 Entered Medline: 19970311

AB Transcription factor E4TF1 is the human homolog of GABP and has been
 renamed hGABP (**human GABP**). hGABP is composed of two
 types of subunits; hGABP betal/E4TF1-53 and the ets-related protein hGABP
 alpha/E4TF1-60. Both bind together to form an (alpha)2(betal)2
 heterotetrameric complex on DNA and activate transcription at specific
 promoters in vitro. Tetramer formation depends on two regions of hGABP
 betal; the N-terminal region containing the Notch/ankyrin-type repeats is
 necessary for binding to hGABP alpha and the C-terminal region is
 necessary for homodimerization. In this report, we constructed various
 deletion mutants of hGABP betal in order to delimit the functional regions
 required for nuclear localization and transcription activity. We found
 that hGABP betal localization in the nucleus is dependent on a region
 located between amino acids 243 and 330 and that the presence of hGABP
 betal influences the efficiency of hGABP alpha transport into the nucleus.
 Next, we demonstrated that the hGABP complex composed of alpha and betal
 subunits activates transcription from the adenovirus early 4 promoter in
 vivo. This transcription activation needs the C-terminal region of hGABP
 betal and is consistent with results obtained with the in vitro assay.
 Furthermore, site-directed mutagenesis analysis of the C-terminal region
 reveals that the alpha-helix structure and the leucine residues are
 important for formation of a heterotetrameric complex with hGABP alpha in
 vitro and for transcription activation in vivo. These results suggest that
 hGABP betal stimulates transcription as part of a heterotetrameric complex
 with hGABP alpha in vivo.

=>